Electrocardiographic “Peri-Infarction Block”

A Clinical and Pathologic Correlation

By C. Hilmon Castle, M.D., and William M. Keane

The term “peri-infarction block” was suggested by First, Bayley, and Bedford1 in 1950 to describe a type of intraventricular conduction defect that develops following myocardial infarction. They described alteration in the direction of the early portion of the QRS complex (initial QRS forces) that produced definite manifestations of myocardial infarction (i.e., Q waves) and slurring and prolongation of the terminal portion of the QRS complex (terminal QRS forces), giving rise to a total QRS duration of 0.11 second or greater. The authors postulated that the normal radial spread of excitation to the epicardium was interrupted by subendocardial infarction and that the overlying active myocardium was activated through circuitous routes of slower conducting myocardial fibers.

Grant and associates2-7 proposed different criteria for “peri-infarction block,” which are (1) an abnormality of the direction of initial QRS forces of a type characteristic of myocardial infarction (accounting for Q waves); (2) an abnormality of direction of terminal QRS forces so that they point in a direction away from the initial QRS forces, the angle between the direction of the initial and terminal forces being 100° or more; (3) little or no prolongation of the QRS interval. These criteria represent a substantial departure from those proposed by First et al.1

Grant interpreted the defect in excitation of the left ventricle in “peri-infarction block” to represent involvement by infarction or scarring of one or the other of the two divisions of the left bundle. In this circumstance impulse conduction is presumed to spread entirely through the uninterrupted division of the bundle with considerable alteration in the sequence of depolarization but with little prolongation of the duration of excitation. Location of the two branches, the superior division along the anterolateral endocardium and the inferior division along the diaphragmatic endocardium is given as the explanation for the two clinical types of “peri-infarction block”: anterolateral and diaphragmatic.

The directions of the mean initial 0.04-second and terminal 0.04-second QRS forces are said to be specific for the location of the infarction. Loss of electrical activity of the subendocardial layer results in the initial QRS forces pointing directly away from the infarct and alterations in the direction of excitation of myocardial fibers in the area of the infarct late to be invaded results in the terminal QRS forces pointing directly toward the infarct. For example, in anterolateral “peri-infarction block” the mean initial 0.04 QRS force points rightward and inferiorly producing a Q wave in leads I and aVf, and the mean terminal 0.04 QRS force points leftward and superiorly producing an S wave in leads II, III, and aVF (figs. 1 and 3). In diaphragmatic peri-infarction block, the mean initial 0.04 QRS force points leftward and superiorly producing Q waves in leads II, III, and aVF and the mean terminal 0.04 QRS force points rightward and inferiorly producing S waves in leads I and aVL (fig. 2).

The diagnostic implications of Grant’s7 postulations are obvious and have been generally accepted. The following survey attempts to define the diagnostic significance of “peri-infarction block” on a statistical basis.

Electrocardiograms (approximately 2,500) of adult patients dying in the Salt Lake General Hospital between 1950 and 1962 were

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Figure 1

Standard 12-lead electrocardiogram from patient with myocardial infarction and anterolateral "peri-infarction block." I₀₄ is initial and T₀₄ is terminal 0.04-second portions of the QRS complex.

Figure 2

Standard 12-lead electrocardiogram from patient with myocardial infarction and diaphragmatic "peri-infarction block." I₀₄ is initial and T₀₄ is terminal 0.04-second portions of the QRS complex.
reviewed for left ventricular conduction defects. "Peri-infarction block" according to Grant's criteria was found on electrocardiograms of 105 patients. Of these, 55 had complete postmortem examinations and clinical records available for review. Nearly all patients were in the older age range (41 to 95 years) with an average age of 70 years. Pathologic examinations were conducted by full-time faculty in the Department of Pathology of the University of Utah College of Medicine. Careful analysis was made of heart weights and thickness of ventricular walls. Coronary arteries and the myocardium were examined by gross and histologic methods. Special attempts to identify the conduction system in the left ventricle were not made.

Of 55 patients with detailed pathologic examinations of their hearts, 44 (80 per cent) had some type of left ventricular disease

### Table 1

<table>
<thead>
<tr>
<th>Analysis of Postmortem Findings in 55 Patients with &quot;Peri-infarction Block.&quot; Average Age 70 Years (Range 41 to 95 Years)</th>
<th>Total</th>
<th>QRS interval &lt; 0.10 sec.</th>
<th>QRS interval &gt; 0.10 sec.</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVH with diffuse scarring</td>
<td>15</td>
<td>9</td>
<td>6</td>
</tr>
<tr>
<td>Gross discrete scar (&gt;1 cm.) in LV from previous infarction</td>
<td>12</td>
<td>8</td>
<td>4</td>
</tr>
<tr>
<td>Diffuse scarring and concomitant severe coronary atherosclerosis but no LVH</td>
<td>11</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>LVH—no scarring</td>
<td>3</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Infiltrative process in LV</td>
<td>3</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Normal LV—mild to moderate atherosclerosis but no enlargement or scarring</td>
<td>11</td>
<td>11</td>
<td>0</td>
</tr>
</tbody>
</table>
(table 1). Only 12 (22 per cent) had discrete myocardial scars due to previous infarction. In 38, however, significant diffuse scarring was present (69 per cent). Three had left ventricular hypertrophy only. In three others an infiltrative process in the myocardium was the only abnormality found: one had metastatic bronchogenic carcinoma and two had marked fatty infiltration. Eleven patients had no gross or microscopic abnormalities in the left ventricles; however, all but one of these had mild to moderate coronary atherosclerosis and chronic obstructive airway disease with emphysema. One patient had no abnormalities of the coronary arteries, heart, or lungs (table 2). He was a 50-year-old man with carcinoma of the pancreas and staphylococcal septicemia. How long the “peri-infarction block” pattern had been present could not be determined since an earlier tracing was not available (fig. 3).

The width of the QRS complex had no value in separating the various types of left ventricular disease, but the QRS interval was normal (<0.10 second) in the 11 patients without evidence of left ventricular disease.

Postmortem examinations were not available for 50 of the 105 patients in this study. Clinical records and evaluations for heart and lung disease were adequate for analysis in 41. Definite evidence for heart disease with involvement of the left ventricle was found in 34 of 41 records reviewed (83 per cent) (table 3). At least one of the following was present in each of 34 patients: a clinical syndrome typical of myocardial infarction, chronic hypertensive vascular disease, valvular (mitral or aortic) heart disease, enlargement of the left ventricle or congestive heart failure.

| Table 2 |
| Analysis of 11 Patients with Normal Left Ventricle on Pathologic Examination and “Peri-infarction Block” on Electrocardiogram. Average Age 69 Years (Range 49 to 92 Years) |
|------------------|------------------|------------------|
| Chronic obstructive airway disease and emphysema | 10 |
| No clinical history or pathologic evidence for heart or lung disease | 1 |

Only five (12 per cent) had experienced a typical myocardial infarction. Of seven patients without evidence for one of the above, one patient had equivocal evidence of enlargement of the heart, three had severe obstructive airway disease and three had no signs, symptoms or x-ray findings that suggested heart or lung disease.

**Discussion**

Ventricular conduction defect following myocardial infarction was described in 1917 by Oppenheimer and Rothschild. The defect was ascribed to interruption of small terminal fibers in the Purkinje network and was called “arborization block.” Their criteria for this type of intraventricular block which consist of prolonged, notched, and low amplitude QRS complexes were found to be of no value in distinguishing various myocardial processes. Similar lesions and mechanism of development were incriminated in the original description of “peri-infarction block;” however, subsequent modification of the criteria and assumptions concerning the mechanism for “peri-infarction block” have been accepted without adequate confirmation by clinical or pathologic studies.

Durrer et al. has demonstrated alteration

| Table 3 |
| Retrospective Clinical Evaluation of 41 Deceased Patients with “Peri-infarction Block” and no Postmortem Examination |
|------------------|------------------|------------------|
| Definite evidence for left ventricular disease | 34 (83%) | 20 | 14 |
| Suggestive evidence for cardiomegaly | 1 | 1 | 0 |
| Chronic obstructive airway disease with emphysema | 3 | 3 | 0 |
| No evidence for heart or lung disease | 3 | 3 | 0 |
of excitation in the left ventricle of dogs with experimentally induced myocardial infarction and scars. The alteration occurs as a result of disturbance in conduction in myocardial fibers in or surrounding the scar. Conduction velocity and the sequence of excitation in the Purkinje fibers were normal in all instances. Excitation of normal myocardium overlying a scar did not necessarily prolong the QRS interval even though it occurred through a circuitous route of myocardial fibers. These studies support the explanation proposed by First, Bayley, and Bedford for the mechanism of intraventricular conduction in myocardial infarction but do not support Grant's hypothesis of "peri-infarction block."

Development of an intraventricular conduction defect following a myocardial infarction is not uncommon; however, the diagnostic significance of "peri-infarction block" as an isolated electrocardiographic finding has not been established. O'Reilly and Sokolow evaluated 19 patients with "peri-infarction block" and found 84 per cent with clinical evidence of heart disease, yet in only 58 per cent did the evidence support coronary artery disease. Kossmann and associates commented on a pathologic-electrocardiographic correlation study at the University of Cincinnati in which "almost all" patients with "peri-infarction block" had anatomic abnormalities of the heart (principally left ventricular hypertrophy and scarring). Approximately 50 per cent had discrete infarctions.

In our evaluation of 96 patients, a high correlation of left ventricular disease and "peri-infarction block" by Grant's criteria was confirmed. Its identification, therefore, is of clinical usefulness. The implication that it is pathognomonic of myocardial infarction is not justified. The variety of anatomic lesions found at necropsy emphasizes the nonspecificity of "peri-infarction block." There is no convincing evidence that it is any more meaningful in identifying heart disease than other types of intraventricular conduction defects involving the left ventricle. Not infrequently, it occurs in association with chronic obstructive airway disease and emphysema without gross or microscopic abnormalities in the myocardium. Also, "peri-infarction block" has been observed in the apparent absence of both heart and lung disease.

Despite the fact that the term "peri-infarction block" has already gained popularity, its implication is misleading and its use therefore should be discouraged. The term is appropriate only when the abnormality occurs in association with other evidence of a myocardial infarction and even then "post-infarction block" would be the preferred term. In those cases of electrocardiographic abnormality without other evidence of myocardial infarction, it is suggested that the defect be identified as an intraventricular conduction defect of the "peri-infarction block type." Since it is not likely that use of this term will be discontinued, we recommend restrictions be placed on its use and efforts to gain a better understanding of the mechanism of development and its significance continue.

Summary

Detailed analysis for the presence and type of heart disease was made in 96 deceased patients who had electrocardiographic "peri-infarction block." Evidence for disease of the left ventricle was found in 80 per cent of these patients. Left ventricular hypertrophy and diffuse scarring of the left ventricle were the most common abnormalities on pathologic examination. Definite myocardial infarction was found in only 12 of the 55 patients (22 per cent) subjected to postmortem examination. Electrocardiographic "peri-infarction block" was therefore not specific for myocardial infarction although it was often associated with it. Use of the term should be restricted to left intraventricular conduction defect of the "peri-infarction block type."

In spite of the nonspecificity of this type of intraventricular conduction defect, it can be a useful clinical sign. If chronic obstructive airway disease and emphysema can be excluded, the presence of some type of left ventricular disease is almost assured.
References


Science and Elegance

The experiments adduced by Dr. Franklin in support of his hypothesis were most ingeniously contrived and happily executed. A singular felicity of induction guided all his researches, and by very small means he established very grand truths. The style and manner of his publication are almost as worthy of admiration as the doctrines it contains. He has endeavoured to remove all mystery and obscurity from the subject; he has written equally for the uninitiated and for the philosopher; and he has rendered his details amusing as well as perspicuous—elegant as well as simple.—Sir Humphry Davy. Quoted by E. N. da C. Andrade, in The scientific work of Benjamin Franklin, Nature 177, 61 (1956).
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